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The Effects of Trisomic Dyrk1a on Ts65Dn Embryonic Craniofacial Development

Emily Haley[1], Mariyamou Diallo[1], Randall J. Roper[1]
[1]Department of Biology, School of Science

Down syndrome (DS) is caused by Trisomy 21 in humans and leads to distinctive craniofacial features in all affected individuals. The Ts65Dn mouse model of DS has orthologs of about half of the genes found on chromosome 21 and mirrors craniofacial phenotypes seen in DS including a small dysmorphic mandible. Previous studies have shown that the small mandible is due to deficits in proliferation and migration from the neural tube of neural crest cell (NCC) craniofacial precursors. *Dyrk1a* is a trisomic gene found in humans with DS and Ts65Dn mice, and it is overexpressed in the 1st pharyngeal arch (PA1) of our mouse model. We hypothesize that Ts65Dn, *Dyrk1a*$^{+/-}$ embryos (otherwise trisomic with the normal 2 copies of *Dyrk1a*) will show similar PA1 and overall embryo size as well as NCC number when compared to euploid littermates. To test our hypothesis we bred Ts65Dn, *Dyrk1a*$^{+/-}$ mothers to generate the following genotypes; Ts65Dn, Eu, Ts65Dn, *Dyrk1a*$^{+/-}$ and Eu, *Dyrk1a*$^{+/-}$. At E9.5 embryos were removed from mothers. Using unbiased stereology on sectioned E9.5 embryos, we measured PA1 NCC number and total embryonic volume. We propose that if Ts65Dn, *Dyrk1a*$^{+/-}$ embryos show a normalized PA1, future work should be concentrated in viable therapies to target overexpression of Dyrk1a in the Ts65Dn mouse model.

Mentor: Randall J. Roper, Department of Biology, IUPUI School of Science, Indiana University-Purdue University Indianapolis